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Case Report

Fulminant Compressive Optic Neuropathy due to Worsening Thyroid Eye Disease after Iodinated Contrast Load: The Jod-Basedow Effect

Rachel, Scott, NitHat, Shruthi Harish Bindiganavile, Amina, Malik, Andrew Lee*
Department of Ophthalmology, Blanton Eye Institute, Houston Methodist Hospital, Houston

***Corresponding author:** Andrew G, Department of Blanton Eye Institute, Houston Methodist Hospital, Houston

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Abstract

Thyroid disease, while common, is also a challenging disease that can present in a variety of ways and often requires close collaboration between general practitioners and a variety of specialists to manage. Here we present the case a 59-year-old man with hypertension and diabetes who presented to the emergency department with chest pain as well as palpitations, tachycardia, and weight loss. Weeks after evaluation with Computed Tomography (CT) of his chest with iodined contrast, he developed worsening peri-orbital edema and acute painful vision loss of the right eye. He was found to have compressive optic neuropathy secondary to worsening thyroid eye disease, and required urgent surgical decompression. This case illustrates the importance of early diagnosis of thyroid eye disease and the need for careful selection of imaging modalities, particularly with regard to the use of contrast, in patients with thyroid disease.

Keywords: Thyroid Eye Disease; Iodine; Iodinated Contrast; Imaging Study Selection; Compressive Optic Neuropathy

Introduction

The thyroid gland produces thyroid hormones including Triiodothyronine (T3) and Thyroxine (T4). Iodine is critical to thyroid hormone production and feedback mechanisms exist to regulate thyroid hormone production based upon serum iodine levels. When there is excess iodine, it is transported via the sodium-iodide symporter into the thyroid, transiently inhibiting thyroid peroxidase, decreasing thyroid hormone synthesis, and leading to the downregulation of the symporter; this is followed by "escape" once iodine levels return below a threshold with normalization of T3/T4 levels (Wolff-Chaikoff effect). [1] However, in patients with thyroid dysregulation (e.g., Graves disease, autonomous thyroid nodules), this negative feedback mechanism is impaired and, rather than transient hypothyroidism, worsening hyperthyroidism may develop as iodine essentially adds fuel to the fire (Jod-Basedow effect).

This worsening thyroid function can lead to worsening of Thyroid Eye Disease (TED). Although worsening of TED and Compressive Optic Neuropathy (CON) have been reported previously after exogenous iodinated contrast, to our knowledge this is the most aggressive, rapid, and severe case of worsening CON due to presumed Jod Basedow effect to be reported in the English language ophthalmic literature.

Case Presentation

A 59-year-old Nigerian man with hypertension and diabetes mellitus presented to the Emergency Department (ED) with intermittent sub-sternal chest pain radiating to the right arm. He also reported palpitations, tachycardia, a 30 pound weight loss, and "swollen" eyelids. Cardiac work-up was unremarkable. Additional work-up revealed critically low Thyroid Stimulating Hormone (TSH) <0.01 and elevated free T4 (>5.6). Computed Tomography (CT) of the chest with contrast revealed enlarged thyroid with multiple nodules. Thyroid uptake scan was deferred, as he had received iodinated contrast, and he was presumed to have toxic multinodular goiter, and discharged on methimazole and propranolol. Two weeks after discharge, endocrinology noted peri-orbital edema and he was treated with a short course of low-dose oral steroids. He saw an outside optometrist who noted elevated Intra Ocular Pressure (IOP) in the 40 mm Hg range and started him on oral acetazolamide and topical anti-glaucoma therapy. He then presented to the ED with worsening peri-orbital edema and a two-day history of acute, painful loss of vision in the right eye (OD).

Ophthalmic exam showed bilateral conjunctival injection, periorbital edema, and exophthalmos. His visual acuity was No Light Perception (NLP) OD and 20/20 in the left eye (OS). He had a right exotropia of 15 prism diopters (PD) and markedly restricted ductions in all gazes consistent with a global bilateral ophthalmoplegia. The right pupil was amaurotic with a dense Relative Afferent Pupillary Defect (RAPD). Slit lamp exam showed 2-3+ chemosis OU and mild corneal edema OD. Ophthalmoscopy showed a Cup To Disc Ratio (CDR) of 0.5 OD and 0.7 OS. IOP measured 37 mm Hg OD and 35 mm Hg OS. Optical Coherence Tomography (OCT) revealed retinal nerve fiber layer loss and diffuse optic atrophy OD, and was unremarkable in OS. Automated perimetry (Humphrey visual field 24-2) showed inferior arcuate depression OS. A non-contrast CT of the orbits showed marked generalized bilateral enlargement of the extraocular muscles with significant crowding in the right orbital apex region consistent with thyroid compressive optic neuropathy OD. (Figure 1 and 2). Intravenous methylprednisolone (1000 mg)

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treatment and an urgent bilateral orbital decompression, with three- wall decompression (medial, lateral and floor) OD and two-wall decompression (medial and floor) OS was performed. At 6 weeks post-operatively, his vision remained NLP OD and was stable OS. There was persistent periorbital edema, but improvement in exophthalmos and conjunctival injection and the IOP was well-controlled with topical anti-glaucoma therapy.

Discussion

CON is a feared complication of Thyroid Eye Disease (TED) and occurs due to orbital apex crowding. This can occur even in the absence of marked proptosis, inflammatory signs, or diplopia. Early detection and treatment, including IV steroids, prompt surgical decompression, and adjunctive low-dose orbital radiotherapy, are crucial to saving vision [2]. Despite aggressive IV steroids and prompt surgical decompression this patient did not recover vision. Interestingly, this patient had evidence for TED during the first outside hospital admission. The administration of the iodinated CT contrast load is hypothesized to have caused dramatic worsening of the TED and secondary rapid and severe CON to NLP OD. The average amount of free iodine after even a single dose of contrast for CT scan is 13,500 μg. This is approximately ten times the recommended daily dose of iodine [3]. The typical thyroid gland response to such a large bolus of iodine is the Wolff-Chaikoff effect, in which large dose of iodine produces a transient inhibition of organic binding of iodide resulting in a mild inhibition of hormone synthesis resolving within 10 days. However, iatrogenic hyperthyroidism can result, particularly in those with impaired thyroid autoregulation-a result known as the Jod-Basedow Effect [1].

There are numerous reports of iodinated contrast induced hyperthyroidism. One patient with history of subclinical hyperthyroidism following iodinated contrast CT 4 years prior developed overt hyperthyroidism 20 weeks after a rechallenge with iodinated contrast CT [4]. Unlike the Wolff-Chaikoff effect, the Jod-Basedow effect often has a variable time of onset and the duration between IV contrast exposure to hyperthyroidism onset is often 2 to 12 weeks or longer [5].

A case-control study by Rhee et al matching new hyperthyroid cases to euthyroid controls, found that iodinated contrast media exposure was associated with development of new hyperthyroidism in a significant number of patients [6]. In another case, a patient with stable, inactive Graves' disease who was status post radioactive thyroid ablation presented to ophthalmology for elective orbital decompression for correction of proptosis. Two weeks after pre-operative contrast CT, she developed ocular pain and worsening proptosis and was found to have CON bilaterally due to TED [3].

In summary, we report a case of fulminant CON due to TED following iodinated contrast in a chest CT. Clinicians should be aware of the possibility of Jod Basedow effect in hyperthyroid patients receiving iodinated contrast even if the imaging is for non-ophthalmic indications. In addition, in typical TED, orbital CT imaging does not require contrast material to visualize the distinctive radiographic finding of extraocular muscle enlargement.

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